PALM INTRANET

Day : Thursday
Date: 11/27/2003

Time: 14:11:29

# Inventor Name Search

Enter the **first few letters** of the Inventor's Last Name.
Additionally, enter the **first few letters** of the Inventor's First name.

| **Last Name** | **First Name** | |
|---|---|---|
| Vinson | Charles | Search |

To go back use Back button on your browser toolbar.

Back to PALM | ASSIGNMENT | OASIS | Home page

| Set Name side by side | Query | Hit Count | Set Name result set |
|---|---|---|---|
| *DB=USPT,PGPB,JPAB,EPAB,DWPI,TDBD; THES=ASSIGNEE; PLUR=YES; OP=AND* | | | |
| L8 | (acidically adj modified) same (N-terminal adj extension) | 2 | L8 |
| L7 | L6 not L5 | 13 | L7 |
| L6 | L4 and (transgenic adj (mouse or mammal)) | 18 | L6 |
| L5 | L4 same (transgenic adj (mouse or mammal)) | 5 | L5 |
| L4 | (Fos or c-Fos) same (dominant adj negative) | 67 | L4 |
| L3 | (4heptaFos) | 0 | L3 |
| L2 | (transgenic adj (mouse or mammal)) same (4heptaFos) | 0 | L2 |
| L1 | Vinson-charles-R$.in. | 4 | L1 |

END OF SEARCH HISTORY

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### Status: Path 1 of [Dialog Information Services via Modem]

### Status: Initializing TCP/IP using (UseTelnetProto 1 ServiceID pto-dialog)
Trying 31060000009999...Open

DIALOG INFORMATION SERVICES
PLEASE LOGON:
 ******** HHHHHHHH SSSSSSSS?
### Status: Signing onto Dialog
 ********
ENTER PASSWORD:
 ******** HHHHHHHH SSSSSSSS? ********
Welcome to DIALOG
### Status: Connected


Dialog level 03.05.00D

Last logoff:  24nov03 17:17:59
Logon file001 27nov03 14:17:19
            *** ANNOUNCEMENT ***
                      ***
--File 654 - US published applications from March 15, 2001 to the
present are now online.  Please see HELP NEWS 654 for details.

                      ***
--File 581 - The 2003 annual reload of Population Demographics is
complete. Please see Help News581 for details.


                     ***
--File 990 - NewsRoom now contains February 2003 to current records.
File 992 - NewsRoom 2003 archive has been newly created and contains
records from January 2003. The oldest months's records roll out of
File 990 and into File 992 on the first weekend of each month.
To search all 2003 records BEGIN 990, 992, or B NEWS2003, a new
OneSearch category.
                      ***
--Connect Time joins DialUnits as pricing options on Dialog.
See HELP CONNECT for information.
                      ***
                      ***
--SourceOne patents are now delivered to your email inbox
as PDF replacing TIFF delivery. See HELP SOURCE1 for more
information.
                      ***
--Important news for public and academic
libraries.  See HELP LIBRARY for more information.
                      ***
--Important Notice to Freelance Authors--
See HELP FREELANCE for more information
                      ***
NEW FILES RELEASED
***Emergency Room (File 454), Hospital Inpatient Profiles (File 462),
   and Hospital Outpatient Profiles (File 463)
***World News Connection (File 985)
***Dialog NewsRoom - 2003 Archive (File 992)
***TRADEMARKSCAN-Czech Republic (File 680)
***TRADEMARKSCAN-Hungary (File 681)
***TRADEMARKSCAN-Poland  (File 682)
                      ***
UPDATING RESUMED
                      ***
RELOADED
***Population Demographics -(File 581)
***CLAIMS Citation (Files 220-222)
```

```
REMOVED
                    ***

      >>> Enter BEGIN HOMEBASE for Dialog Announcements <<<
      >>>    of new databases, price changes, etc.       <<<
                    ****
KWIC is set to 50.
HILIGHT set on as '*'
* * *                                    * * *

File   1:ERIC  1966-2003/Nov 20
       (c) format only 2003 The Dialog Corporation

      Set  Items  Description
      ---  -----  -----------
Cost is in DialUnits
?b 155, 159, 5, 73
       27nov03 14:17:35 User259876 Session D568.1
            $0.31    0.088 DialUnits File1
     $0.31  Estimated cost File1
     $0.06  TELNET
     $0.37  Estimated cost this search
     $0.37  Estimated total session cost   0.088 DialUnits


SYSTEM:OS  - DIALOG OneSearch
  File 155:MEDLINE(R)  1966-2003/Nov W3
        (c) format only 2003 The Dialog Corp.
*File 155: Medline has temporarily stopped updating with
Completed records (Nov 2003). Please see HELP NEWS 154 for details.
  File 159:Cancerlit  1975-2002/Oct
        (c) format only 2002 Dialog Corporation
*File 159: Cancerlit ceases updating with immediate effect.
Please see HELP NEWS.
  File   5:Biosis Previews(R)  1969-2003/Nov W4
        (c) 2003 BIOSIS
*File   5: BIOSIS Previews has been reloaded with major enhancements.
See HELP NEWS005 for more information.
  File  73:EMBASE  1974-2003/Nov W4
        (c) 2003 Elsevier Science B.V.

      Set  Items  Description
      ---  -----  -----------
?s (transgenic (w) (mouse or mice or mammal)) (s) (4heptaFos)
         137826  TRANSGENIC
        1556264  MOUSE
        1547215  MICE
         136221  MAMMAL
              0  4HEPTAFOS
      S1      0  (TRANSGENIC (W) (MOUSE OR MICE OR MAMMAL)) (S)
                 (4HEPTAFOS)
?s (Fos or c-Fos) (s) ((dominant (w) negative) or truncated)
          59522  FOS
           7084  C-FOS
         265596  DOMINANT
        1489594  NEGATIVE
          35767  DOMINANT(W)NEGATIVE
         213992  TRUNCATED
      S2   1978  (FOS OR C-FOS) (S) ((DOMINANT (W) NEGATIVE) OR TRUNCATED)
?s s2 (s) (transgenic)
           1978  S2
         137826  TRANSGENIC
      S3     65  S2 (S) (TRANSGENIC)
?rd
...examined 50 records  (50)
...completed examining records
```

```
      S4       23  RD (unique items)
?s s4 and (knockout)
              23  S4
           63459  KNOCKOUT
      S5        2  S4 AND (KNOCKOUT)
?t s5/3,k/all
```

**5/3,K/1 (Item 1 from file: 155)**

DIALOG(R)File 155:MEDLINE(R)


11909029 99352251 PMID: 10421778

**A dominant-negative mutant of c-Jun inhibits cell cycle progression during the transition of CD4(-)CD8(-) to CD4(+)CD8(+) thymocytes.**

King L B; Tolosa E; Lenczowski J M; Lu F; Lind E F; Hunziker R; Petrie H T; Ashwell J D

Laboratory of Immune Cell Biology, National Cancer Institute, National Institutes of Health, Bethesda MD 20892, USA.

International immunology (ENGLAND) Aug 1999, 11 (8) p1203-16, ISSN 0953-8178 Journal Code: 8916182

Contract/Grant No.: AI 33940; AI; NIAID; CA09162; CA; NCI

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

... to be necessary for many TCR-mediated events in mature T cells, relatively little is known about their roles in thymocyte development. We have generated *transgenic* mice that express a trans-*dominant*-*negative* mutant of c-Jun (TAM-67) specifically in thymocytes. Expression of TAM-67 inhibited the up-regulation of AP-1-responsive genes such as c-jun and IL-2 in stimulated thymocytes from *transgenic* mice. In addition, altered thymocyte development in TAM-67-expressing mice was revealed by a decrease in thymic cellularity ( approximately 50%) which could be accounted...

... decrease in the number of CD4(-)CD8(-)CD25(-) cells in the S + G(2)/M stages of the cell cycle. These results indicate that Jun/*Fos*-containing transcription factors promote the proliferative burst that accompanies the transition from the CD4(-)CD8(-) to the CD4(+)CD8(+) stage of thymocyte development.

...; Cycle; Cell Differentiation; Gene Expression Regulation, Developmental; Interleukin-2--genetics--GE; Interleukin-2--metabolism--ME; Lymphocyte Activation; Mice; Mice, Inbred C57BL; Mice, Inbred DBA; Mice, *Knockout*; Mice, Transgenic; Proto-Oncogene Proteins c-jun--genetics--GE; Receptors, Interleukin-2--metabolism--ME; Thymus Gland--embryology--EM; Transcription Factor AP-1--genetics--GE; Transcription...

**5/3,K/2 (Item 2 from file: 155)**

DIALOG(R)File 155:MEDLINE(R)


09259792 20576860 PMID: 11134330

**Interleukin-6-induced STAT3 and AP-1 amplify hepatocyte nuclear factor 1-mediated transactivation of hepatic genes, an adaptive response to liver injury.**

Leu J I; Crissey M A; Leu J P; Ciliberto G; Taub R

Department of Genetics, University of Pennsylvania School of Medicine, Philadelphia, Pennsylvania 19104, USA.

Molecular and cellular biology (UNITED STATES) Jan 2001, 21 (2) p414-24, ISSN 0270-7306 Journal Code: 8109087

Contract/Grant No.: DK19629; DK; NIDDK; DK49210; DK; NIDDK; P30 DK50306; DK; NIDDK

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

... mechanisms. Evidence for a biologic role of IL-6 in IGFBP-1 upregulation was demonstrated by increased expression of hepatic IGFBP-1 in IL-6 *transgenic* and following injection of IL-6 into nonfasting animals and its reduced expression in IL-6(-/-) livers posthepatectomy. In both hepatic and nonhepatic cells, IL...
... factor 1 (HNF-1) site and was dependent on the presence of endogenous liver factor HNF-1 and induced factors STAT3 and AP-1 (c-*Fos*/c-Jun). IL-6 acted through the STAT3 pathway, as *dominant* *negative* STAT3 completely blocked IL-6-mediated stimulation of the IGFBP-1 promoter via the HNF-1 site. HNF-1/c-*Fos* and HNF-1/STAT3 protein complexes were detected in mouse livers and in hepatic and nonhepatic cell lines overexpressing STAT3/c-*Fos* /HNF-1. Similar regulation was demonstrated using glucose-6-phosphatase and alpha-fibrinogen promoters, indicating that HNF-1/IL-6/STAT3/AP-1-mediated transactivation...
...; Like Growth-Factor Binding Protein 1--metabolism--ME; Interleukin-6 --genetics--GE; Interleukin-6--pharmacology--PD; Liver--drug effects--DE; Liver--metabolism--ME; Mice; Mice, *Knockout*; Precipitin Tests; Promoter Regions (Genetics)--genetics--GE; Protein Binding;. Proto-Oncogene Proteins c-fos--genetics--GE; Proto-Oncogene Proteins c-fos--metabolism--ME; Proto-Oncogene...
?ds

| Set | Items | Description |
|---|---|---|
| S1 | 0 | (TRANSGENIC (W) (MOUSE OR MICE OR MAMMAL)) (S) (4HEPTAFOS) |
| S2 | 1978 | (FOS OR C-FOS) (S) ((DOMINANT (W) NEGATIVE) OR TRUNCATED) |
| S3 | 65 | S2 (S) (TRANSGENIC) |
| S4 | 23 | RD (unique items) |
| S5 | 2 | S4 AND (KNOCKOUT) |

?s s4 not s5

| | | |
|---|---|---|
| | 23 | S4 |
| | 2 | S5 |
| S6 | 21 | S4 NOT S5 |

?t s6/3,k/all

**6/3,K/1 (Item 1 from file: 155)**
DIALOG(R)File 155:MEDLINE(R)


15061859 22592528 PMID: 12706249
**Inducible, brain region-specific expression of a dominant negative mutant of c-Jun in transgenic mice decreases sensitivity to cocaine.**
Peakman M-C; Colby C; Perrotti L I; Tekumalla P; Carle T; Ulery P; Chao J ; Duman C; Steffen C; Monteggia L; Allen M R; Stock J L; Duman R S; McNeish J D; Barrot M; Self D W; Nestler E J; Schaeffer E
Department of Exploratory Medicinal Sciences and CNS Discovery, Pfizer Global Research and Development, Eastern Point Road, Groton, CT 06340, USA.
Brain research (Netherlands) Apr 25 2003, 970 (1-2) p73-86, ISSN 0006-8993 Journal Code: 0045503
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

... members of the Jun family to form active AP-1 transcription factor complexes. In the present study, we took advantage of this property and generated *transgenic* mice, using the tetracycline gene regulation system, that support the inducible, brain region-specific expression of a *dominant* *negative* mutant form of c-Jun (Deltac-Jun), which can antagonize the actions of *Fos* proteins. Expression of Deltac-Jun in the striatum and certain other brain regions of adult mice decreases their development of cocaine-induced conditioned place preference...

**6/3,K/2 (Item 2 from file: 155)**
DIALOG(R)File 155:MEDLINE(R)

14639133 22474043 PMID: 12586760

**c-Jun N-terminal kinase activation of activator protein-1 underlies homologous regulation of the gonadotropin-releasing hormone receptor gene in alpha T3-1 cells.**

Ellsworth Buffy S; White Brett R; Burns Ann T; Cherrington Brian D; Otis Annette M; Clay Colin M

Animal Reproduction and Biotechnology Laboratory, Department of Biomedical Sciences, College of Veterinary Medicine and Biomedical Sciences, Colorado State University, Fort Collins, Colorado 80523, USA.

Endocrinology (United States) Mar 2003, 144 (3) p839-49, ISSN 0013-7227 Journal Code: 0375040

Contract/Grant No.: HD-08558-01; HD; NICHD; HD-32416; HD; NICHD

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

... 3)). Herein we find that selective removal of the canonical AP-1 site leads to a loss of GnRH regulation of the GnRHR promoter in *transgenic* mice. Thus, an intact AP-1 element is necessary for GnRH responsiveness of the GnRHR gene both in vitro and in vivo. Based on in vitro analyses, GnRH appeared to enhance the interaction of JunD, FosB, and c-*Fos* at the GnRHR AP-1 element. Although enhanced binding of cFos reflected an increase in gene expression, GnRH appeared to regulate both FosB and JunD...

... induced ERK activation eliminated the GnRH response of the GnRHR promoter. GnRH responsiveness was, however, lost in alpha T3-1 cells that stably express a *dominant*-*negative* c-Jun N-terminal kinase (JNK) kinase, suggesting a critical role for JNK in mediating GnRH regulation of the GnRHR gene. Consistent with this possibility...

**6/3,K/3 (Item 3 from file: 155)**

DIALOG(R)File 155:MEDLINE(R)



11441926 98324933 PMID: 9658179

**Osteoblastic responses to TGF-beta during bone remodeling.**

Erlebacher A; Filvaroff E H; Ye J Q; Derynck R

Departments of Growth and Development, University of California at San Francisco, San Francisco, California 94143, USA.

Molecular biology of the cell (UNITED STATES) Jul 1998, 9 (7) p1903-18, ISSN 1059-1524 Journal Code: 9201390

Contract/Grant No.: AR-41126; AR; NIAMS; DE-10306; DE; NIDCR; GM07618; GM ; NIGMS

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

... bone resorption by osteoclasts; however, the molecular basis of these inductive interactions is unknown. We have previously shown that osteoblastic overexpression of TGF-beta2 in *transgenic* mice deregulates bone remodeling and leads to an age-dependent loss of bone mass that resembles high-turnover osteoporosis in humans. This phenotype implicates TGF...

...gain insight into the physiological role of TGF-beta in bone remodeling, we have now characterized the responses of osteoblasts to TGF-beta in these *transgenic* mice. We took advantage of the ability of alendronate to specifically inhibit bone resorption, the lack of osteoclast activity in c-*fos*-/- mice, and a new *transgenic* mouse line that expresses a *dominant*-*negative* form of the type II TGF-beta receptor in osteoblasts. Our results show that TGF-beta directly increases the steady-state rate of osteoblastic differentiation...

**6/3,K/4 (Item 4 from file: 155)**
DIALOG(R)File 155:MEDLINE(R)


10884842 97236666 PMID: 9121774

**A role for the small GTPase Rac in polyomavirus middle-T antigen-mediated activation of the serum response element and in cell transformation.**

Urich M; Senften M; Shaw P E; Ballmer-Hofer K
Friedrich Miescher-Institute, Basel, Switzerland.

Oncogene (ENGLAND) Mar 13 1997, 14 (10) p1235-41, ISSN 0950-9232
Journal Code: 8711562
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

...as model systems to study mitogenic signaling and cell transformation. These proteins stimulate cell growth in cultured cells and induce tumors in virus infected or *transgenic* animals. One of these proteins, polyomavirus middle-T, acts like a constitutively activated tyrosine growth factor receptor. Middle-T recruits several cellular enzymes into a...

...S phase. Our data show that Rho family GTPases play an essential role in cell transformation by middle-T. Furthermore, we demonstrate that the c-*fos* promoter is activated by two Ras-initiated signaling cascades. One is Raf-dependent and requires binding of SHC and PI 3-kinase to the middle-T complex. This pathway signals via ternary complex factor (TCF) to the serum response element (SRE) of the c-*fos* promoter. Signaling to TCF by Raf also depends on functional Rac, but not CDC42, as demonstrated in luciferase reporter assays with an ETS domain-containing...

... pathway is Raf-independent, does not require SHC but functional PI 3-kinase, and transduces signals via Rac to serum response factor (SRF). Microinjection of *dominant* *negative* Rac1 blocks nuclear translocation of ERK1 in middle-T-expressing cells. This lends support to the idea that the two signaling cascades initiated by Ras...

**6/3,K/5 (Item 5 from file: 155)**
DIALOG(R)File 155:MEDLINE(R)


10872437 97224076 PMID: 9070657

**Overexpression of the FosB2 gene in thymocytes causes aberrant development of T cells and thymic epithelial cells.**

Carrozza M L; Jacobs H; Acton D; Verma I; Berns A
Division of Molecular Genetics of the Netherlands Cancer Institute, Amsterdam.

Oncogene (ENGLAND) Mar 6 1997, 14 (9) p1083-91, ISSN 0950-9232
Journal Code: 8711562
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

We have examined the role of the AP-1 transcription factor on thymocyte maturation and thymus architecture by overexpressing FosB2 in *transgenic* mice. FosB2 is a naturally occurring splice variant of the FosB2 gene, encoding a *truncated* protein which lacks two domains necessary for transcriptional activation. The expression of FosB2 in the thymocytes severely affected their maturation and the structure of the...

...marrow derived cells, as it could be reproduced in bone marrow chimaeric mice. This pathology was very reminiscent to that observed in mice overexpressing c-*Fos* in thymic epithelium: also in that case the thymus underwent with age a progressive expansion of the epithelium and major changes in the ratio of thymocyte subsets, but the phenotype appeared to be

an intrinsic property of the epithelial cells since it could not be reproduced by *transgenic* bone marrow transplantation. We speculate that both overexpression of FosB2 in thymocytes and overexpression of c-*Fos* in thymic epithelium results in aberrant signaling between thymocytes and stroma, that ultimately alters the thymic micromilieu, leading to this severe pathology.

**6/3,K/6 (Item 6 from file: 155)**
DIALOG(R)File 155:MEDLINE(R)


10332839 96135139 PMID: 8538746

**Defective thymocyte proliferation and IL-2 production in transgenic mice expressing a dominant-negative form of CREB.**

Barton K; Muthusamy N; Chanyangam M; Fischer C; Clendenin C; Leiden J M
Department of Medicine, University of Chicago, Illinois 60637, USA.

Nature (ENGLAND) Jan 4 1996, 379 (6560) p81-5, ISSN 0028-0836 Journal Code: 0410462
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

... resting thymocytes contain predominantly unphosphorylated (inactive) CREB, which is rapidly activated by phosphorylation on Ser 119 following thymocyte activation. T-cell development is normal in *transgenic* mice that express a *dominant*-*negative* form of CREB (CREBA119, with alanine at position 119) under the control of the T-cell-specific CD2 promoter/enhancer. In contrast, thymocytes and T...

...apoptotic death in response to a number of different activation signals. This proliferative defect is associated with the markedly reduced induction of c-jun, c-*fos*, Fra-2 and FosB following activation of the CREBA119 *transgenic* thymocytes. We propose that T-cell activation leads to the phosphorylation and activation of CREB, which in turn is required for normal induction of the...

**6/3,K/7 (Item 7 from file: 155)**
DIALOG(R)File 155:MEDLINE(R)


10275824 96077615 PMID: 8528457

**Cerebral energy metabolism and immediate early gene induction following severe incomplete ischaemia in transgenic mice overexpressing the human ornithine decarboxylase gene: evidence that putrescine is not neurotoxic in vivo.**

Lukkarainen J; Kauppinen R A; Koistinaho J; Halmekyto; Alhonen L M; Janne J
NMR Research Group, A.I. Virtanen Institute, Kuopio, Finland.

European journal of neuroscience (ENGLAND) Sep 1 1995, 7 (9) p1840-9, ISSN 0953-816X Journal Code: 8918110
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

... decarboxylase followed by accumulation of putrescine, and these biochemical phenomena have been thought to contribute to the development of neuronal damage. We have used a *transgenic* mouse line overexpressing the human ornithine decarboxylase gene in their neurons with constitutively high putrescine to study the possible role of putrescine in development of ...
... of approximately 7.1 to approximately 6.5 and an increase in lactate concentration from < 1 to approximately 10 mmol/kg in both syngenic and *transgenic* mice. Following deocclusion, recovery of energy metabolites

intracellular pH and lactate were identical in both animal groups. Ornithine decarboxylase activity rose 9- and 3-fold in syngenic and *transgenic* mice respectively 6 h after ischaemia, which was approximately 50-fold greater than the basal level in syngenic mice. In situ hybridization experiments revealed induction of transcription factors c-*Fos* and zif-268 in the hippocampus, throughout the cerebral cortex and striatum 1-3 h after ischaemia. Messenger RNA of heat shock protein 70 was induced in dentate gyrus and CA3 and CA4 subfields of the hippocampus 1 h after ischaemia.(ABSTRACT *TRUNCATED* AT 250 WORDS)

**6/3,K/8 (Item 8 from file: 155)**
DIALOG(R)File 155:MEDLINE(R)


10100714 22060504 PMID: 12065418
**The basis for TCR-mediated regulation of the IL-2 receptor alpha chain gene: role of widely separated regulatory elements.**
Kim Hyoung-Pyo; Leonard Warren J
Laboratory of Molecular Immunology, Building 10, Room 7N252, National Heart, Lung, and Blood Institute, National Institutes of Health, Bethesda, MD 20892, USA. hpkim@helix.nih.gov
EMBO journal (England) Jun 17 2002, 21 (12) p3051-9, ISSN 0261-4189 Journal Code: 8208664
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

... 2 responsiveness requires Stat5 and HMG-I(Y) binding, TCR responsiveness of PRRIV requires two AP-1- and two NFAT-binding sites that bind Jun, *Fos* and NFAT family members in vitro and in vivo. Moreover, IL-2Ralpha induction is impaired in T lymphocytes from *transgenic* mice expressing a *dominant*-*negative* c-jun construct, or following treatment with cyclosporin A. Thus, our data indicate an important role for both AP-1 and NFAT proteins for TCR...

**6/3,K/9 (Item 9 from file: 155)**
DIALOG(R)File 155:MEDLINE(R)


09667571 21457312 PMID: 11572966
**DeltaFosB: a sustained molecular switch for addiction.**
Nestler E J; Barrot M; Self D W
Department of Psychiatry and Center for Basic Neuroscience, University of Texas Southwestern Medical Center, 5323 Harry Hines Boulevard, Dallas, TX 75390-9070, USA. eric.nestler@utsouthwestern.edu
Proceedings of the National Academy of Sciences of the United States of America (United States) Sep 25 2001, 98 (20) p11042-6, ISSN 0027-8424 Journal Code: 7505876
Document type: Journal Article; Review; Review, Tutorial
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

... one mechanism by which drugs of abuse produce relatively stable changes in the brain that contribute to the addiction phenotype. DeltaFosB, a member of the *Fos* family of transcription factors, accumulates within a subset of neurons of the nucleus accumbens and dorsal striatum (brain regions important for addiction) after repeated administration...

... DeltaFosB represents a molecular mechanism that could initiate and then sustain changes in gene expression that persist long after drug exposure ceases. Studies in inducible *transgenic* mice that overexpress either DeltaFosB or a *dominant* *negative* inhibitor of the protein provide direct evidence that DeltaFosB causes increased sensitivity to the

behavioral effects of drugs of abuse and, possibly, increased drug seeking ...

**6/3,K/10 (Item 10 from file: 155)**
DIALOG(R)File 155:MEDLINE(R)


09667140 21456836 PMID: 11572782
**An important role of neural activity-dependent CaMKIV signaling in the consolidation of long-term memory.**
Kang H; Sun L D; Atkins C M; Soderling T R; Wilson M A; Tonegawa S
Howard Hughes Medical Institute, New Haven, CT 06511, USA.
Cell (United States) Sep 21 2001, 106 (6) p771-83, ISSN 0092-8674 Journal Code: 0413066
Contract/Grant No.: R01-NS32925; NS; NINDS; RO1-NS27037; NS; NINDS
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

... CaMKIV) has been implicated in the regulation of CRE-dependent transcription. To investigate the role of this kinase in neuronal plasticity and memory, we generated *transgenic* mice in which the expression of a *dominant*-*negative* form of CaMKIV (dnCaMKIV) is restricted to the postnatal forebrain. In these *transgenic* mice, activity-induced CREB phosphorylation and c-*Fos* expression were significantly attenuated. Hippocampal late LTP (L-LTP) was also impaired, whereas basic synaptic function and early LTP (E-LTP) were unaffected. These deficits...

**6/3,K/11 (Item 11 from file: 155)**
DIALOG(R)File 155:MEDLINE(R)


09661531 21450721 PMID: 11566906
**Angiotensin II type 2 receptor inhibits epidermal growth factor receptor transactivation by increasing association of SHP-1 tyrosine phosphatase.**
Shibasaki Y; Matsubara H; Nozawa Y; Mori Y; Masaki H; Kosaki A; Tsutsumi Y; Uchiyama Y; Fujiyama S; Nose A; Iba O; Tateishi E; Hasegawa T; Horiuchi M; Nahmias C; Iwasaka T
Department of Medicine II, Kansai Medical University, Moriguchi, Osaka, Japan.
Hypertension (United States) Sep 2001, 38 (3) p367-72, ISSN 1524-4563 Journal Code: 7906255
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

... shown to inactivate ERK, the action of AT(2) on EGFR activation remains undefined. Using AT(2)-overexpressing vascular smooth muscle cells from AT(2) *transgenic* mice, we studied these undefined actions of AT(2). Maximal ERK activity induced by Ang II was increased 1.9- and 2.2-fold by ...
...2) stimulation and association of SHP-1 with EGFR was increased, whereas AT(2) failed to tyrosine phosphorylate SHP-1. Stable overexpression of SHP-1-*dominant* *negative* mutant completely abolished AT(2)-mediated inhibition of EGFR and ERK activation. AT(1)-mediated c-*fos* mRNA accumulation was attenuated by 48% by AT(2) stimulation. Induction of fibronectin gene containing an AP-1 responsive element in its 5'-flanking region...

**6/3,K/12 (Item 12 from file: 155)**
DIALOG(R)File 155:MEDLINE(R)

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09465047 21238511 PMID: 11340164

**Tissue-specific *transgenic* knockdown of *Fos*-related antigen 2 (Fra-2) expression mediated by *dominant* *negative* Fra-2.**

Smith M; Burke Z; Humphries A; Wells T; Klein D; Carter D; Baler R

School of Bioscience, Cardiff University, Cardiff, United Kingdom.

Molecular and cellular biology (United States) Jun 2001, 21 (11) p3704-13, ISSN 0270-7306 Journal Code: 8109087

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

**Tissue-specific *transgenic* knockdown of *Fos*-related antigen 2 (Fra-2) expression mediated by *dominant* *negative* Fra-2.**

... as does the basis of its selective effects on transcriptional activity. To pursue these issues, we created a transgenic rat line (NATDNF2) in which a *dominant* *negative* fra-2 (DNF2) gene is strongly expressed in the pineal gland; tissue selectivity was achieved by putting the DNF2 gene under the control of the...

**6/3,K/13 (Item 13 from file: 155)**

DIALOG(R)File 155:MEDLINE(R)

(c) format only 2003 The Dialog Corp. All rts. reserv.

09426549 21194221 PMID: 11299744

**Transactivation of AP-1 in AP-1-luciferase reporter transgenic mice by arsenite and arsenate.**

Huang C; Bode A M; Chen N Y; Ma W Y; Li J; Nomura M; Dong Z

Hormel Institute, University of Minnesota, 801 16th Avenue NE, Austin, MN 55912, USA.

Anticancer research (Greece) Jan-Feb 2001, 21 (1A) p261-7, ISSN 0250-7005 Journal Code: 8102988

Contract/Grant No.: CA74916; CA; NCI; CA81064; CA; NCI

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

... kinases are proposed to be responsible for the tumor promotion activity by arsenic. Induction of AP-1 DNA binding activity and c-jun and c-*fos* expression was reported to be only observed in cells responding to arsenite, but not to arsenate. However, in this study, we found that both arsenite...

...kinases and protein kinase C because increased AP-1 activity by arsenite could be blocked by either treatment of cells with PD98059 or overexpression of *dominant* *negative* protein kinase Ca. Furthermore, both arsenite and arsenate could induce transactivation of AP-1 in AP-1-luciferase reporter *transgenic* mice.

**6/3,K/14 (Item 14 from file: 155)**

DIALOG(R)File 155:MEDLINE(R)

(c) format only 2003 The Dialog Corp. All rts. reserv.

08904888 20192021 PMID: 10725720

**Analysis of signals and functions of the chimeric human granuloctye-macrophage colony-stimulating factor receptor in BA/F3 cells and transgenic mice.**

Watanabe S; Aoki Y; Nishijima I; Xu M; Arai K

Department of Molecular Biology, Institute of Medical Science, University of Tokyo, Tokyo, Japan. sumiko@ims.u-tokyo.ac.jp

Journal of immunology (Baltimore, Md. - 1950) (UNITED STATES) Apr 1 2000, 164 (7) p3635-44, ISSN 0022-1767 Journal Code: 2985117R

Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

... phosphorylation of Jak1 but not of Jak2 occurred with stimulation of hGM-CSF, the dominant-negative Jak2 but not the dominant-negative Jak1 *suppresses c-*fos* promoter activation*. To determine whether the chimeric receptor alpha/beta,beta is functional in vivo, we developed *transgenic* mice expressing the chimeric receptor alpha/beta,beta. Bone marrow cells from the *transgenic* mice expressing the alpha/beta,beta receptor form not only GM colonies but also various lineages of colonies in response to GM-CSF. In addition, mast cells were produced when bone marrow cells of the *transgenic* mouse were cultured with hGM-CSF. Thus, it appears that the cytoplasmic region of the alpha subunit is not required for hGM-CSF promoting activities...

**6/3,K/15 (Item 15 from file: 155)**
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2003 The Dialog Corp. All rts. reserv.

08591078 95279392 PMID: 7759507
**Impaired insulin signaling in skeletal muscles from transgenic mice expressing kinase-deficient insulin receptors.**
Chang P Y; Goodyear L J; Benecke H; Markuns J S; Moller D E
Charles A. Dana Research Institute, Boston, Massachusetts, USA.
Journal of biological chemistry (UNITED STATES) May 26 1995, 270 (21) p12593-600, ISSN 0021-9258 Journal Code: 2985121R
Contract/Grant No.: R29-AR42238; AR; NIAMS; RO1 45874-01; PHS
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

*Transgenic* mice which overexpress kinase-deficient human insulin receptors in muscle were used to study the relationship between insulin receptor tyrosine kinase and the in vivo...

... *downstream signaling pathways. Intravenous insulin stimulated insulin* receptor tyrosine kinase activity by 7-fold in control muscle versus < or = 1.5-fold in muscle from *transgenic* mice. Similarly, insulin failed to stimulate tyrosyl phosphorylation of receptor beta-subunits or insulin receptor substrate 1 (IRS-1) in *transgenic* muscle. Insulin substantially stimulated IRS-1-associated phosphatidylinositol (PI) 3-kinase in control versus absent stimulation in *transgenic* muscles. In contrast, insulin-like growth factor 1 modestly stimulated PI 3-kinase in both control and *transgenic* muscle. The effects of insulin to stimulate p42 mitogen-activated protein kinase and c-*fos* mRNA expression were also markedly impaired in *transgenic* muscle. Specific immunoprecipitation of human receptors followed by measurement of residual insulin receptors suggested the presence of hybrid mouse-human heterodimers. In contrast, negligible hybrid formation involving insulin-like growth factor 1 receptors was evident. We conclude that (i) *transgenic* expression of kinase-defective insulin receptors exerts *dominant*-*negative* effects at the level of receptor auto-phosphorylation and kinase activation; (ii) insulin receptor tyrosine kinase activity is required for in vivo insulin-stimulated IRS-1 phosphorylation, IRS-1-associated PI 3-kinase activation, phosphorylation of mitogen-activated protein kinase, and c-*fos* gene induction in skeletal muscle; (iii) hybrid receptor formation is likely to contribute to the in vivo *dominant*-*negative* effects of kinase-defective receptor expression.

**6/3,K/16 (Item 16 from file: 155)**
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2003 The Dialog Corp. All rts. reserv.

07672742 93127988 PMID: 8420113

**A fos-lac Z transgenic mouse that can be used for neuroanatomic mapping.**

Smeyne R J; Schilling K; Oberdick J; Robertson L; Luk D; Curran T; Morgan J I

Department of Neuroscience, Roche Institute of Molecular Biology, Roche Research Center, Nutley, New Jersey 07110.

Advances in neurology (UNITED STATES) 1993, 59 p285-91, ISSN 0091-3952 Journal Code: 0367524

Document type: Journal Article; Review; Review, Tutorial

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

... further the utility of this approach, a bacterial gene encoding beta-galactosidase (lac Z) has been fused, in frame, into the fourth exon of c-*fos*, and this *fos*-lac Z fusion gene has been introduced into the germ line of mice. We have analyzed the expression of beta-galactosidase (under the control of the c-*fos* promoter) in the developing and adult nervous systems of these *transgenic* mice. As far as can be determined, the constitutive and stimulated expression of the transgene accurately reflects the expression of cognate c-*fos* in both cultured cells and the intact animal. This study has also revealed novel sites of constitutive and induced expression of c-*fos* that were overlooked using conventional analysis. In particular, constitutive expression of c-*fos* is associated with cells that are entering terminal differentiation and are destined to die. In addition, induced expression of the transgene in adult brain mirrors the pattern of neurotoxicity elicited by kainic acid.(ABSTRACT *TRUNCATED* AT 250 WORDS)

**6/3,K/17 (Item 1 from file: 159)**

DIALOG(R)File 159:Cancerlit



02143179 PMID: 96600463

**The role of transcription factors in tumor promotion: Potential molecular targets for drugs, dietary modulation or gene prevention (Meeting abstract).**

Colburn; Dong; Watts; Bernstein

Laboratory of Viral Carcinogenesis, NCI-FCRDC, Frederick, MD 21702-1201

Non-serial 1994, 2nd Denver Conference on Nutrition and Cancer, September 7-11, 1994, Denver, CO 1994.,

Document Type: JOURNAL ARTICLE

Languages: ENGLISH

Main Citation Owner: NOTNLM

Record type: Completed

... AP-1 transcription factor activity revealed activation by TPA or EGF in P+ cells but not in P- cells. AP-1, composed of Jun and *Fos* family dimers, drives the expression of a number of genes including those encoding various proteases and DNA tumor virus genes. To address the question of...

... inhibitors. The first included retinoids and glucocorticoids whose receptors, apparently, bind to Jun and inactivate its transcription factor activity. The second was to express a *dominant* *negative* Jun mutant (TAM67) that is transcriptionally inactive and forms complexes with endogenous Jun and *Fos* , rendering them transcriptionally inactive. Retinoid and glucocorticoids exert many AP-1-independent effects, including those mediated by RAR and GR response elements. It was necessary to use a more specific inhibitor such as TAM67 (*dominant* *negative* Jun). When TAM67 was expressed in eight clonal transfectants, all were blocked for both tumor promoter-induced AP-1 activation and induced transformation, thus suggesting...

... Current studies are aimed at assessing the role of induced AP-1 in certain human carcinogenesis cell culture models as well as in vivo in

*transgenic* mice.

**6/3,K/18 (Item 1 from file: 5)**
DIALOG(R)File 5:Biosis Previews(R)


0014347646 BIOSIS NO.: 200300305135
**REGULATION OF GENE EXPRESSION IN THE NUCLEUS ACCUMBENS BY DELTAFOSB AND CREB.**
AUTHOR: McClung C A (Reprint); Nestler E J (Reprint)
AUTHOR ADDRESS: Dept of Psychiatry, UT Southwestern Med Ctr, Dallas, TX, USA**USA
JOURNAL: Society for Neuroscience Abstract Viewer and Itinerary Planner 2002 pAbstract No. 502.11 2002 2002
MEDIUM: cd-rom
CONFERENCE/MEETING: 32nd Annual Meeting of the Society for Neuroscience Orlando, Florida, USA November 02-07, 2002; 20021102
SPONSOR: Society for Neuroscience
DOCUMENT TYPE: Meeting; Meeting Poster; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

...ABSTRACT: of both AP1 complexes (composed of Fos and Jun proteins) and of CREB, since many of these genes are downregulated in mice expressing either a *dominant* *negative* form of CREB (mCREB) or cJun (DELTAcJun). Interestingly, several of the genes that are upregulated by DELTAFosB after 1-2 weeks of expression are downregulated...

**6/3,K/19 (Item 2 from file: 5)**
DIALOG(R)File 5:Biosis Previews(R)


0013777473 BIOSIS NO.: 200200370984
**Differential role for CREB transcription factor in regulation of B-1 and B-2 B cell development in mice**
AUTHOR: Chen Hui-Chen (Reprint); Muthusamy Natarajan
AUTHOR ADDRESS: Molecular Virology, Immunology and Medical Genetics, Ohio State University, 700 Children's Drive, Columbus, OH, 43205, USA**USA
JOURNAL: FASEB Journal 16 (5): pA1246 March 22, 2002 2002
MEDIUM: print
CONFERENCE/MEETING: Annual Meeting of Professional Research Scientists on Experimental Biology New Orleans, Louisiana, USA April 20-24, 2002; 20020420
ISSN: 0892-6638
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

...ABSTRACT: function. Consistant with thishypothesis, overexpression of a non-phosphorylatable dominant negative form of CREB-1 in bone marrow resulted in B cell developmental defects in *transgenic* mice. Thus, the mutant CREB *transgenic* mice revealed dramatically reduced IgM/B220 positive mature B-2 cells in the spleen and bone marrow with significant increase in peritoneal B1-B cells. CFDS analysis of B1 cells revealed normal growth/proliferation properties of B1 cell in *transgenic* mice. Multicolor FACS analysis identified an early block at pre-BI to Pre-BII transition stage in the bone marrow. Further analysis indicated that this blockage is independent of Bcl2 or BclxL. RT-PCR analysis revealed the upregulation of c-*Fos* and JunB in *transgenic* mice pre-BII cells. This model will be used to define the molecular basis of CREB transcription factor in regulation of B-1 and B...

**6/3,K/20 (Item 1 from file: 73)**
DIALOG(R)File 73:EMBASE

10649341 EMBASE No: 2000114310

**Analysis of signals and functions of the chimeric human granulocyte-macrophage colony-stimulating factor receptor in BA/F3 cells and transgenic mice**

Watanabe S.; Aoki Y.; Nishijima I.; Xu M.-J.; Arai K.-I.
Dr. S. Watanabe, Dept. of Molec./Developmental Biol., University of Tokyo, 4-6-1 Shirokanedai, Minato-ku, Tokyo 108-8639 Japan
AUTHOR EMAIL: sumiko@ims.u-tokyo.ac.jp

Journal of Immunology ( J. IMMUNOL. ) (United States) 01 APR 2000, 164/7 (3635-3644)
CODEN: JOIMA ISSN: 0022-1767
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 35

...phosphorylation of Jak1 but not of Jak2 occurred with stimulation of hGM-CSF, the dominant-negative Jak2 but not the dominant- negative Jak1 suppresses c-*fos* promoter activation. To determine whether the chimeric receptor alpha/beta,beta is functional in vivo, we developed *transgenic* mice expressing the chimeric receptor alpha/beta,beta. Bone marrow cells from the *transgenic* mice expressing the alpha/beta,beta receptor form not only GM colonies but also various lineages of colonies in response to GM-CSF. In addition, mast cells were produced when bone marrow cells of the *transgenic* mouse were cultured with hGM-CSF. Thus, it appears that the cytoplasmic region of the alpha subunit is not required for hGM-CSF promoting activities...

**6/3,K/21 (Item 2 from file: 73)**
DIALOG(R)File 73:EMBASE


07781905 EMBASE No: 1999265128

**A dominant-negative mutant of c-Jun inhibits cell cycle progression during the transition of CD4sup -CD8sup - to CD4sup +CD8sup + thymocytes**

King L.B.; Tolosa E.; Lenczowski J.M.; Lu F.; Lind E.F.; Hunziker R.; Petrie H.T.; Ashwell J.D.
L.B. King, University of Pennsylvania, School of Medicine, 415 Curie Boulevard, Philadelphia, PA 19104 United States

International Immunology ( INT. IMMUNOL. ) (United Kingdom) 1999, 11/8 (1203-1215)
CODEN: INIME ISSN: 0953-8178
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 66

...to be necessary for many TCR-mediated events in mature T cells, relatively little is known about their roles in thymocyte development. We have generated *transgenic* mice that express a trans-*dominant*-*negative* mutant of c-Jun (TAM-67) specifically in thymocytes. Expression of TAM-67 inhibited the up-regulation of AP-1-responsive genes such as c-jun and IL-2 in stimulated thymocytes from *transgenic* mice. In addition, altered thymocyte development in TAM-67-expressing mice was revealed by a decrease in thymic cellularity (~ 50%) which could be accounted for...

...decrease in the number of CD4sup -CD8sup -CD25sup - cells in the S + Ginf 2/M stages of the cell cycle. These results indicate that Jun/*Fos* -containing transcription factors promote the proliferative burst that accompanies the transition from the CD4sup -CD8sup - to the CD4sup +CD8sup + stage of thymocyte development.
?ds

| Set | Items | Description |
|---|---|---|
| S1 | 0 | (TRANSGENIC (W) (MOUSE OR MICE OR MAMMAL)) (S) (4HEPTAFOS) |
| S2 | 1978 | (FOS OR C-FOS) (S) ((DOMINANT (W) NEGATIVE) OR TRUNCATED) |

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S3          65   S2 (S) (TRANSGENIC)
S4          23   RD (unique items)
S5           2   S4 AND (KNOCKOUT)
S6          21   S4 NOT S5
?s s3 and (adipose (w) tissue)
              65  S3
          103031  ADIPOSE
         2652742  TISSUE
           95140  ADIPOSE(W)TISSUE
      S7       0  S3 AND (ADIPOSE (W) TISSUE)
?s (c/EBP) (s) ((dominant (w) negative) or knockout or truncated)
>>>Term "EBP" is not defined in one or more files
         3133184  C/EBP
          265596  DOMINANT
         1489594  NEGATIVE
           35767  DOMINANT(W)NEGATIVE
           63459  KNOCKOUT
          213992  TRUNCATED
      S8   59409  (C/EBP) (S) ((DOMINANT (W) NEGATIVE) OR KNOCKOUT OR
                  TRUNCATED)
?s s8 (s) (transgenic)
           59409  S8
          137826  TRANSGENIC
      S9    1312  S8 (S) (TRANSGENIC)
?s s9 and (adipose (w) tissue)
            1312  S9
          103031  ADIPOSE
         2652742  TISSUE
           95140  ADIPOSE(W)TISSUE
     S10      14  S9 AND (ADIPOSE (W) TISSUE)
?rd
...completed examining records
     S11       5  RD (unique items)
?t s11/3,k/all
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**11/3,K/1 (Item 1 from file: 155)**

DIALOG(R)File 155:MEDLINE(R)
(c) format only 2003 The Dialog Corp. All rts. reserv.

11571372 99003070 PMID: 9784492

**Life without white fat: a transgenic mouse.**

Moitra J; Mason M M; Olive M; Krylov D; Gavrilova O; Marcus-Samuels B; Feigenbaum L; Lee E; Aoyama T; Eckhaus M; Reitman M L; Vinson C

Laboratory of Biochemistry, National Cancer Institute (NCI), National Institutes of Health (NIH), Bethesda, Maryland 20892 USA.

Genes & development (UNITED STATES) Oct 15 1998, 12 (20) p3168-81, ISSN 0890-9369 Journal Code: 8711660

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

We have generated a *transgenic* mouse with no white fat tissue throughout life. These mice express a *dominant*-*negative* protein, termed A-ZIP/F, under the control of the adipose-specific aP2 enhancer/promoter. This protein prevents the DNA binding of B-ZIP transcription factors of both the *C*/EBP and Jun families. The *transgenic* mice (named A-ZIP/F-1) have no white *adipose* *tissue* and dramatically reduced amounts of brown *adipose* *tissue* , which is inactive. They are initially growth delayed, but by week 12, surpass their littermates in weight. The mice eat, drink, and urinate copiously, have...

Descriptors: *Adipose* *Tissue*--abnormalities--AB; *Mice, Transgenic --genetics--GE

**11/3,K/2 (Item 2 from file: 155)**

DIALOG(R)File 155:MEDLINE(R)

11356514 98236893 PMID: 9575979

**Temperature-dependent feeding: lack of role for leptin and defect in brown *adipose* *tissue*-ablated obese mice.**

Melnyk A; Himms-Hagen J
Department of Biochemistry, University of Ottawa, Ontario, Canada.

American journal of physiology (UNITED STATES) Apr 1998, 274 (4 Pt 2) pR1131-5, ISSN 0002-9513 Journal Code: 0370511
Contract/Grant No.: DK-46930; DK; NIDDK
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

**Temperature-dependent feeding: lack of role for leptin and defect in brown *adipose* *tissue*-ablated obese mice.**

The objective was to characterize the ability of control and *transgenic* brown *adipose* *tissue* (BAT)-ablated uncoupling protein diphtheria toxin A chain (UCP-DTA) mice to adjust food intake in relation to changes in environmental temperature and to assess...

... involvement of leptin in this adjustment. We measured serum leptin in mice from a previous study of UCP-DTA mice raised at thermoneutrality (35 degrees *C*) or at the usual rearing temperature (24 degrees *C*) from weaning [Melnyk, A., M. -E. Harper, and J. Himms-Hagen. Am. J. Physiol, 272 (Regulatory Integrative Comp. Physiol. 41): R1088-R1093, 1997] and extended the study by acclimating control and obese UCP-DTA mice at 18 wk of age to cold (14 degrees *C*) for up to 14 days. Leptin levels did not change in control mice at 14 degrees *C*; however, food intake increased threefold within 1 day and remained at this level. Serum leptin level was elevated in UCP-DTA mice at 24 degrees *C* compared with control mice at 24 degrees *C* ; this elevated level decreased within 1 day at 14 degrees *C* and was not different from the level in control mice by 14 days. Food intake of UCP-DTA mice that were hyperphagic at 24 degrees *C* did not change during 7 days at 14 degrees *C*, then increased slowly. Similar low leptin levels were present in control mice raised at 24 or 35 degrees *C* and in UCP-DTA mice raised at 35 degrees *C*. Food intake of control mice raised at 24 degrees *C* was two times that of control mice raised at 35 degrees *C*. UCP-DTA mice raised at 35 degrees *C* ate the same low amount as control mice raised at 35 degrees *C*. UCP-DTA mice at 24 degrees *C* were hyperphagic relative to control mice at 24 degrees *C* yet had elevated leptin levels in their serum. Two principal conclusions are drawn. First, adjustment of food intake over a fourfold range by control mice acclimated to temperatures from 35 down to 14 degrees *C* is independent of changes in serum leptin levels. Second, this adjustment of food intake in relation to temperature is defective in the UCP-DTA mouse; the defect leads to hyperphagia at 24 degrees *C* and a failure to increase food intake as rapidly as control mice when exposed to 14 degrees *C*. Because lack of UCP-1-mediated thermogenesis in BAT of *knockout* mice is known not to induce hyperphagia, we propose that deficiency of UCP-1-expressing brown adipocytes in BAT of UCP-DTA mice results in...

**11/3,K/3 (Item 3 from file: 155)**
DIALOG(R)File 155:MEDLINE(R)


09731871 21532924 PMID: 11606718

**C/EBPalpha is required for differentiation of white, but not brown, *adipos * *tissue*.**

Linhart H G; Ishimura-Oka K; DeMayo F; Kibe T; Repka D; Poindexter B; Bick R J; Darlington G J
Department of Gastroenterology/Hepatology, University Hospital Freiburg, Hugstetter Strasse 55, 79106 Freiburg, Germany.

Proceedings of the National Academy of Sciences of the United States of America (United States) Oct 23 2001, 98 (22) p12532-7, ISSN 0027-8424

Journal Code: 7505876
Contract/Grant No.: DK45285; DK; NIDDK
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

**C/EBPalpha is required for differentiation of white, but not brown, *adipose* *tissue*.**

The transcription factor CCAAT enhancer binding protein alpha (*C* /EBPalpha) is expressed at high levels in liver and *adipose* *tissue*. Cell culture studies show that *C* /EBPalpha is sufficient to trigger differentiation of preadipocytes into mature adipocytes, suggesting a central role for *C*/EBPalpha in the development of *adipose* *tissue*. *C* /EBPalpha *knockout* mice die within 7-12 h after birth. Defective gluconeogenesis of the liver and subsequent hypoglycemia contribute to the early death of these animals. This short life span impairs investigation of the development of *adipose* *tissue* in these mice. To improve the survival of *C*/EBPalpha-/- animals, we generated a *transgenic* line that expresses *C*/EBPalpha under the control of the albumin enhancer/promoter. This line was bred into the *knockout* strain to generate animals that express *C* /EBPalpha in the liver but in no other tissue. The presence of the transgene improved survival of *C*/EBPalpha-/- animals almost 3-fold. *Transgenic* *C*/EBPalpha-/- animals at 7 days of age show an absence of s. *c*., perirenal, and epididymal white fat despite excess lipid substrate in the serum, whereas brown *adipose* *tissue* is somewhat hypertrophied and shows minimal biochemical alterations. Interestingly, mammary gland fat tissue is present and exhibits normal morphology. The absence of white *adipose* *tissue* in many depots in the presence of high serum lipid levels shows that *C*/EBPalpha is required for the in vivo development of this tissue. In contrast, brown *adipose* *tissue* differentiation is independent of *C*/EBPalpha expression. The presence of lipid in brown *adipose* *tissue* serves as an internal nutritional control, indicating that neither nutritional intake nor lipoprotein composition is likely responsible for the absence of white fat.

Descriptors: *Adipose* *Tissue*--cytology--CY; *Brown Fat--cytology--CY; *CCAAT-Enhancer-Binding Protein-alpha--physiology--PH

**11/3,K/4 (Item 4 from file: 155)**
DIALOG(R)File 155:MEDLINE(R)


09670503 21460372 PMID: 11576996

**Role of the high mobility group A proteins in human lipomas.**

Fedele M; Battista S; Manfioletti G; Croce C M; Giancotti V; Fusco A

Dipartimento di Biologia e Patologia Cellulare e Molecolare, Facolta di Medicina e Chirurgia, Universita degli Studi di Napoli, Via Pansini, 5, I-80131 Naples, Italy.

Carcinogenesis (England) Oct 2001, 22 (10) p1583-91, ISSN 0143-3334
Journal Code: 8008055
Document type: Journal Article; Review; Review, Academic
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

... lipomas. 12q13-15 chromosomal translocations involving the HMGA2 gene locus, account for these rearrangements. The HMGA proteins have three AT-hook domains and an acidic *C*-terminal tail. The HMGA2 modifications consist in the loss of the *C* -terminal tail and fusion with ectopic sequences. A pivotal role of the HMGA2 rearrangements in the process of lipomagenesis is suggested by experiments showing that *transgenic* mice carrying a *truncated* HMGA2 gene showed a giant phenotype together with abdominal/pelvic lipomatosis. As HMGA2 null mice showed a great reduction in fat tissue, a positive role...

; Adipocytes--metabolism--ME; *Adipose* *Tissue*--metabolism--ME; Mice; Mice, Transgenic

**11/3,K/5 (Item 5 from file: 155)**
DIALOG(R)File 155:MEDLINE(R)


08971181 20261566 PMID: 10747931

***Transgenic* mice expressing a *truncated* form of the high mobility group I-*C* protein develop adiposity and an abnormally high prevalence of lipomas.**

Arlotta P; Tai A K; Manfioletti G; Clifford C; Jay G; Ono S J
Schepens Eye Research Institute, Division of Rheumatology, Immunology & Allergy, Department of Medicine, Brigham & Women's Hospital, and Committee on Immunology, Harvard Medical School, Boston, Massachusetts 02114, USA.

Journal of biological chemistry (UNITED STATES) May 12 2000, 275 (19) p14394-400, ISSN 0021-9258 Journal Code: 2985121R
Contract/Grant No.: R01 GM49661; GM; NIGMS
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

***Transgenic* mice expressing a *truncated* form of the high mobility group I-*C* protein develop adiposity and an abnormally high prevalence of lipomas.**

Chromosomal translocations in human lipomas frequently create fusion transcripts encoding high mobility group (HMG) I-*C* DNA-binding domains and *C* -terminal sequences from different presumed transcription factors, suggesting a potential role for HMG I-*C* in the development of lipomas. To evaluate the role of the HMG I-*C* component, the three DNA-binding domains of HMG I-*C* have now been expressed in *transgenic* mice. Despite the ubiquitous expression of the *truncated* HMG I-*C* protein, the *transgenic* mice develop a selective abundance of fat tissue early in life, show marked *adipose* *tissue* inflammation, and have an abnormally high incidence of lipomas. These findings demonstrate that the DNA-binding domains of HMG I-*C*, in the absence of a *C*-terminal fusion partner, are sufficient to perturb adipogenesis and predispose to lipomas. We provide data supporting the central utility of this animal model as a...

Descriptors: *Adipose* *Tissue*; *DNA--metabolism--ME; *High Mobility Group Proteins--genetics--GE; *Lipoma--genetics--GE

?ds

| Set | Items | Description |
|---|---|---|
| S1 | 0 | (TRANSGENIC (W) (MOUSE OR MICE OR MAMMAL)) (S) (4HEPTAFOS) |
| S2 | 1978 | (FOS OR C-FOS) (S) ((DOMINANT (W) NEGATIVE) OR TRUNCATED) |
| S3 | 65 | S2 (S) (TRANSGENIC) |
| S4 | 23 | RD (unique items) |
| S5 | 2 | S4 AND (KNOCKOUT) |
| S6 | 21 | S4 NOT S5 |
| S7 | 0 | S3 AND (ADIPOSE (W) TISSUE) |
| S8 | 59409 | (C/EBP) (S) ((DOMINANT (W) NEGATIVE) OR KNOCKOUT OR TRUNCATED) |
| S9 | 1312 | S8 (S) (TRANSGENIC) |
| S10 | 14 | S9 AND (ADIPOSE (W) TISSUE) |
| S11 | 5 | RD (unique items) |

?logoff

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            $2.26    0.706 DialUnits File155
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            $4.83  23 Types
     $7.09  Estimated cost File155
            $0.76    0.259 DialUnits File159
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            $0.26  1 Types
     $1.02  Estimated cost File159
            $4.30    0.767 DialUnits File5
               $3.50  2 Type(s) in Format  3
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            $3.50  2 Types
     $7.80  Estimated cost File5
            $5.58    0.603 DialUnits File73
               $5.10  2 Type(s) in Format  3
            $5.10  2 Types
    $10.68  Estimated cost File73
            OneSearch, 4 files,  2.336 DialUnits FileOS
     $2.32  TELNET
    $28.91  Estimated cost this search
    $29.28  Estimated total session cost   2.424 DialUnits


### Status: Signed Off. (10 minutes)
```